mens for outpatients are the following: (1) Burning of normal skin by higher concentrations necessitates careful application of the stiff paste on the lesions by a skilled nursing staff; (2) anthralin can stain bedclothes and sheets, making home use difficult; (3) prolonged applications are sometimes needed; (4) anthralin should not be used in skin folds, and (5) if anthralin inadvertently gets in the eyes, severe burning may result. These problems have been partially overcome by the newer methods of application discussed below.

Schaefer and co-workers used 1 percent anthralin with 1 percent salicylic acid in petrolatum, which they applied to the psoriatic lesions each day; the ointment was left on for one hour and then removed with soap and water. As far as possible, the anthralin should not be placed on normal appearing skin because this may cause erythema and a burning sensation. It is preferable to follow each treatment with application of an emollient ointment or cream. If this regimen is continued daily, it is successful in clearing 30 percent of the psoriatic lesions and improving an additional 50 percent. The advantage of this method is its short application time, which allows a patient to use anthralin while relaxing at home and avoids staining clothes and sheets. The chief disadvantage is occasional burning that may occur around treated plaques.

In Sweden, Brody and Johansson used 0.01 percent to 0.03 percent anthralin with 10 percent salicylic acid in petrolatum on a daily basis. This ointment does not need to be so carefully applied to lesions only, as burning of normal skin is rarely seen. It usually does not stain clothing so the ointment may be applied in the evening and morning. The main disadvantage is the slow response of lesions, which may take up to three months to resolve.

In England, Seville and co-workers compared the use of 0.25 percent anthralin in a cream base (Dithrocream; dithranol is the generic name) with 0.25 percent anthralin in an ointment base. He found the two preparations to be equally effective. However, patients preferred the easier application of the cream base although it caused more staining than the ointment base.

Anthralin continues to be an important agent in the inhospital treatment of patients with psoriasis. Newer methods of application are making its use possible to outpatients as well.

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Lymphomatoid Papulosis

THE CLINICAL AND HISTOLOGICAL assessment of patients with atypical infiltrates suggestive of lymphoma or leukemia but confined only to the skin has been a difficult problem for many years. In some of these patients involvement of other organs occurs within a short time and a definite diagnosis can be made. In others, however, crops of lesions may continue to develop for many years. These lesions spontaneously resolve within a few weeks and are accompanied by no other evidence of neoplastic disease. The term lymphomatoid papulosis, proposed by Macaulay in 1978, has been the most popular designation for this latter group.

The most common clinical pattern is an inflammatory papular or papulonecrotic eruption that is indistinguishable from pityriasis lichenoides acuta (Mucha-Habermann disease). Mycosis fungoides may be difficult to exclude but clinical evidence of small, short-lived papulonecrotic lesions as well as histological evidence of hemorrhage and epidermal necrosis are said to favor a diagnosis of lymphomatoid papulosis. The relationship of lymphomatoid papulosis to pityriasis lichenoides and, indeed, the nature of pityriasis lichenoides, both remain unclear. It is interesting that in some patients with this clinical pattern the condition responds favorably to ultraviolet light exposure.

Clinical diagnosis is made even more difficult by the fact that some of these patients have papular, plaque-type or nodular lesions that do not resemble pityriasis lichenoides. These lesions generally show less clinical and histological evidence of inflammation and are more suggestive of the tumors of mycosis fungoides or other lymphomas, but still may wax and wane for many years.

Although the great majority of patients reported in the literature have experienced a prolonged benign course, there have been at least three cases in which visceral lymphoma developed and one that has evolved into the more usual pattern of

mycosis fungoides. The significance of the different clinical patterns is not clear at present, but it seems likely that "lymphomatoid papulosis" is not a single disease entity. The application of recently developed immunological methods may help to better define the nature of these perplexing eruptions and their relationship to lymphoid neoplasia.

At present there is no specific therapy for these eruptions and it is clear that these patients should be kept under periodic observation.

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Inflammatory Lesions of Acne Vulgaris: Current Concepts

PATHOGENIC MICROORGANISMS are not commonly present in the inflammatory lesions of acne, and the microorganisms that are present do not invade and multiply in the tissues. Thus, these inflammatory lesions do not involve an infection in the usual sense.

Minor inflammatory lesions are small, very superficial, immediately perifollicular and intrafollicular, short-lived and usually heal without scarring. Such lesions may be benefited by topical therapy. They appear to be the consequence of polymorphonuclear leukocyte aggregation in response to chemotactic factors in the follicle.

Major inflammatory lesions begin deeper (in the dermis), forming nodules of varying size and severity, often persisting for a long time. The crucial event responsible is disruption of the follicle wall with escape of follicle contents into the dermis. The contents include disintegrated epithelial and sebaceous cells, sebum in varying stages of chemical change, microflora including anaerobic *Propionibacterium acnes,* aerobic *Staphylococcus epidermidis,* yeasts, metabolites of these organisms (lipase, proteases and others), free fatty acids, squalene, keratin and hair fragments. Many of these are antigenic and when combined with lipids with adjuvant properties, have the potential for induction of immune reactions. Others can cause primary irritation and foreign body reactions. Thus, the follicle contents include a variety of possible pathogenic agents.

Histologically, the major lesions present elements of mixed granuloma, and infiltrates of polymorphonuclear leukocytes, histiocytes, macrophages and lymphocytes. Fibroblasts and newly formed capillaries are commonly observed. Vasculitis is not present. Hemorrhage and necrosis with ulceration may occur and scarring can be severe.

To prevent the occurrence of major lesions it is necessary to block those mechanisms which disrupt the follicle wall. A frequent finding in early lesions is the invasion of the follicle wall by polymorphonuclear leukocytes with abscess formation. Release of lytic and toxic agents, which may damage the wall, would be a likely consequence. The concept that obstruction of the follicle ostium with subsequent build up of sebum, formation of a comedo, and pressure rupture of the follicle is untenable because it has been shown that disruption occurs in follicles in which obstruction and gross comedones are not present.

In view of the nature of the lesions and the absence of active infection, it is not unexpected that antibacterial agents exert no major beneficial effect on existing inflammatory lesions, and there are no controlled data to show that they do. To the extent that they are effective, it is by decreasing the occurrence of new lesions. Presumably, the mechanism is by reduction of the bacterial population of susceptible follicles.

Existing inflammatory lesions do respond to nonspecific antiinflammatory agents, particularly corticosteroids, either by local or systemic routes of administration. When severe lesions in great numbers are present, systemic therapy is required. In certain cases, for reasons unknown, intensely inflammatory lesions supervene in what had been mild and not otherwise unusual acne vulgaris. The lesions are characterized by pain, hemorrhage, sterile suppuration, gelatinous granulation tissue, liquifying necrosis, ulceration and severe scarring. In some instances the onset has been preceded by viral infections, measles and infectious mononucleosis, or periods of extreme emotional stress. In many cases, no trigger factors are recognized. The nature and pathogenesis of the lesions have not been clarified.

This subject was comprehensively reviewed by Goldschmidt and co-workers in a report in 1977. Systemic corticosteroid therapy was necessary for control. Most cases reverted to their previous state